L Number	Hits	Search Text	DB	Time stamp
2	0	4683202.pn. and label$6 near3 sample	USPAT; US-PGPUB	2003/07/09 08:55
3	12617	label$6 near3 sample	USPAT; US-PGPUB	2003/07/09 08:55
4	1345	label$6 near3 sample near3 (nucleic or DNA)	USPAT; US-PGPUB	2003/07/09 08:57
5	199	(label$6 near3 sample near3 (nucleic or DNA)) same (PCR or polymerase adj1 chain)	USPAT; US-PGPUB	2003/07/09 08:58
6	33	((label$6 near3 sample near3 (nucleic or DNA)) same (PCR or polymerase adj1 chain)) same cDNA	USPAT; US-PGPUB	2003/07/09 08:57
7	7	((label$6 near3 sample near3 (nucleic or DNA)) same (PCR or polymerase adj1 chain)) same cDNA	USPAT	2003/07/09 08:57
8	98	(label$6 near3 sample near3 (nucleic or DNA)) same (PCR or polymerase adj1 chain)	USPAT	2003/07/09 08:59
9	1	4963663.pn.	USPAT	2003/07/09 08:59
1	1	4683202.pn.	USPAT; US-PGPUB	2003/07/09 09:18
10	0	4683202.pn. and cDNA	USPAT; US-PGPUB	2003/07/09 09:18
11	1	4683202.pn. and complementary adj1 dna	USPAT; US-PGPUB	2003/07/09 09:19
12	1	4683202.pn. and sample	USPAT; US-PGPUB	2003/07/09 09:20
13	1	4683202.pn. and label$4	USPAT; US-PGPUB	2003/07/09 09:20

(FILE 'HOME' ENTERED AT 07:19:56 ON 09 JUL 2003)

FILE 'MEDLINE, BIOSIS, CAPLUS, ESBIOBASE, JICST-EPLUS' ENTERED AT 07:20:07 ON 09 JUL 2003

L1 4832 S (17Q22 OR 17Q23 OR 17Q24 OR 17Q21 OR 17Q25 OR 17(2A)(Q21 OR Q
L2 696 S L1 AND AMPLIFI?
L3 256 S L2 AND BREAST (7A) (CANCER? OR CARCINO? OR TUMOR? OR TUMOUR?
L4 12 S L3 AND PY<1992
L5 5 DUP REM L4 (7 DUPLICATES REMOVED)

FILE 'STNGUIDE' ENTERED AT 07:32:06 ON 09 JUL 2003

=>

CS Sussex Centre for Medical Research, University of Sussex, Brighton, UK.
SO BRITISH JOURNAL OF CANCER, **(1989 Oct)** 60 (4) 505-10.
Journal code: 0370635. ISSN: 0007-0920.
CY ENGLAND: United Kingdom
DT Journal; Article; (JOURNAL ARTICLE)
LA English
FS Priority Journals
EM 198912
ED Entered STN: 19900328
Last Updated on STN: 20000303
Entered Medline: 19891215
AB A panel of 73 samples, including 52 primary **breast carcinomas**, 10 normal **breast** tissues and 11 axillary lymph nodes, has been analysed for the presence of **amplifications** and gross structural alterations, in the oncogenes c-erbB-2, c-erbA, c-myc, N-myc, c-mos and c-Ha-ras. The tumours were also classified, graded and staged histopathologically and their DNA ploidy (42 samples) was determined by flow cytometry. Three **breast cancer** cell lines (MCF7, ZR-75-1 and T47D) were also included in the study. **Amplification** of c-erbB-2 was detected in 28% of the tumours, of which 91% had an increased steady-state level of c-erbB-2 mRNA. **Amplification** of c-erbA was found in 23% of tumours and was always associated with the **amplification** of c-erbB-2. Ten out of 12 (83%) tumours which had c-erbB-2 and c-erbA co-**amplification** had metastasised to axillary lymph nodes (P less than 0.006). However, the human thymidine kinase gene, which is present at the same chromosomal location as these two oncogenes (**17q21**-22), was **amplified** in only tw tumours. **Amplification** of c-myc was detected in 21% of the tumours studied, of which 82% (P less than 0.005) were of histopathological grade 3 and none were of grade 1. Flow cytometry showed that 90% (P less than 0.01) of the analysed tumours with c-erbB-2 and c-erbA co-**amplification**, and 70% (P less than 0.1) of those with c-myc **amplification** were DNA aneuploid. This study demonstrates the potential value of c-myc **amplification** in the assessment of the tumour grade, rather than metastatic potential; and of the co-**amplification** of c-erbB-2 and c-erbA as a strong indicator of metastatic potential, rather than tumour grade.

L5 ANSWER 5 OF 5 MEDLINE DUPLICATE 5
AN 89210192 MEDLINE
DN 89210192 PubMed ID: 2707103
TI Selection of cells with different chromosomal localizations of the **amplified** c-myc gene during in vivo and in vitro growth of the **breast carcinoma** cell line SW 613-S.
AU Cherif D; Lavialle C; Modjtahedi N; Le Coniat M; Berger R; Brison O
CS Laboratoire de Cytogenetique, U301 INSERM and UM7 CNRS, Centre Hayem, Hopital Saint-Louis, Paris, France.
SO CHROMOSOMA, **(1989 Jan)** 97 (4) 327-33.
Journal code: 2985138R. ISSN: 0009-5915.
CY GERMANY, WEST: Germany, Federal Republic of
DT Journal; Article; (JOURNAL ARTICLE)
LA English
FS Priority Journals
EM 198906
ED Entered STN: 19900306
Last Updated on STN: 19970203
Entered Medline: 19890606
AB The c-myc gene is **amplified** in the human **breast carcinoma** cell line SW 613-S. At early in vitro passages, the extra copies of the gene were mainly localized in double minute chromosomes (DMs), as shown by in situ hybridization with a biotinylated c-myc probe. However, cells without DMs were also present in which the c-myc genes were found integrated into any of several distinct chromosomes

(mainly 7q+, 4 and 4q+, and 1). When this cell line was propagated in vitro, the level of c-myc **amplification** decreased because cells with DMs and a high **amplification** level were lost and replaced by cells without DMs and having a low **amplification** level. On the contrary, when early passage SW 613-S cells were grown in vivo, as subcutaneous tumours in nude mice, cells with numerous DMs and a high level of c-myc **amplification** were selected for. In one cell line (SW 613-Tu1) established from such a tumour, the DM-containing cells were substituted at late passages for cells with a high number of c-myc copies integrated within an abnormally banded region, at band **17q24** of a 17q+ chromosome. When only cells with integrated genes were present, this cell line was still highly tumorigenic indicating that the localization of the c-myc genes in DMs was not required for these cells to be tumorigenic in nude mice. Furthermore, cells of the secondary tumours induced by SW 613-Tu1 did not contain any DMs showing that in vivo growth did not promote the release of integrated c-myc copies into DMs.

-5 bib ab

L5 ANSWER 1 OF 5 MEDLINE DUPLICATE 1
AN 92034677 MEDLINE
DN 92034677 PubMed ID: 1682035
TI Accumulation of genetic alterations and progression of primary **breast cancer**.
AU Sato T; Akiyama F; Sakamoto G; Kasumi F; Nakamura Y
CS Department of Biochemistry, Cancer Institute, Tokyo, Japan.
SO CANCER RESEARCH, **(1991 Nov 1)** 51 (21) 5794-9.
Journal code: 2984705R. ISSN: 0008-5472.
CY United States
DT Journal; Article; (JOURNAL ARTICLE)
LA English
FS Priority Journals
EM 199111
ED Entered STN: 19920124
Last Updated on STN: 19950206
Entered Medline: 19911125
AB In order to detect common regions of deletion, 219 **breast tumors** were examined for loss of heterozygosity at several loci on chromosomes 3p, 16q, and 17 by restriction fragment length polymorphism analysis. Allelic deletions of loci on chromosomes 3p, 13q, 16q, and 17, and **amplification** of the erbB2 oncogene, were analyzed and compared with histopathological and clinical features. Common regions of deletion were detected within chromosomal bands 3p13-14.3, 16q22-23, 17p13 (two separated loci), and **17q21**. Concordant losses of alleles on chromosomes 3p, 13q, 16q, 17p, and 17q were observed. A significant association was detected between loss of heterozygosity on chromosomes 17p and 17q and **amplification** of the erbB2 oncogene (17p, P = 0.000721, by Fisher's exact test; 17q, P less than 0.001, chi 2 = 12.135). Furthermore, tumors showing highly malignant phenotypes had accumulated more genetic changes at the loci studied than those having less malignant phenotypes on the basis of histopathological classification, lymph node metastasis, and tumor size. These results suggested that accumulation of genetic alterations, including loss of function of tumor suppressor genes on chromosomes 3p, 13q, 16q, and 17, and **amplification** of the erbB2 oncogene, may contribute to **tumor** development and/or progression in primary **breast cancer**.

L5 ANSWER 2 OF 5 MEDLINE DUPLICATE 2
AN 91033775 MEDLINE
DN 91033775 PubMed ID: 1977681
TI The gene for 17 beta-hydroxysteroid dehydrogenase maps to human chromosome **17**, bands q12-**q21**, and shows an RFLP with ScaI.
AU Winqvist R; Peltoketo H; Isomaa V; Grzeschik K H; Mannermaa A; Vihko R
CS Department of Clinical Genetics, Oulu University Central Hospital, Finland.
SO HUMAN GENETICS, **(1990 Oct)** 85 (5) 473-6.
Journal code: 7613873. ISSN: 0340-6717.
CY GERMANY: Germany, Federal Republic of
DT Journal; Article; (JOURNAL ARTICLE)
LA English
FS Priority Journals
EM 199012
ED Entered STN: 19910208
Last Updated on STN: 19950206
Entered Medline: 19901205
AB The gene encoding human 17 beta-hydroxysteroid dehydrogenase (17-HSD; EC 1.1.1.62) is assigned to chromosome 17 by Southern blotting analyses of panels of human x rodent somatic cell hybrids and independently to 17q12-q21 using chromosomal in situ hybridization. A search for physical linkage between 17-HSD and the proto-oncogenes. THRA1 and ERBB2 (both

reported to be located in this region of chromosome 17) was performed by pulsed-field gel electrophoresis (PFGE) using several rare-cutting restriction endonucleases. Because all three genes hybridized to DNA fragments of different lengths it seems unlikely that the gene for 17-HSD is located very close to THRA1 and ERBB2. Further evidence for this assumption was obtained from the absence of any coamplification of the 17-HSD gene in 9 **breast tumors** with **amplification** of the ERBB2 gene. Analyses of Southern blots of ScaI-digested DNAs from unrelated individuals from Northern Finland revealed a relatively infrequent diallelic restriction fragment length polymorphism, the allele frequencies of which were 0.04 (A1) and 0.96 (A2).

L5 ANSWER 3 OF 5 MEDLINE DUPLICATE 3
AN 89248991 MEDLINE
DN 89248991 PubMed ID: 2566377
TI Correlation between long-term survival in **breast cancer** patients and **amplification** of two putative oncogene-coamplification units: hst-1/int-2 and c-erbB-2/ear-1.
AU Tsuda H; Hirohashi S; Shimosato Y; Hirota T; Tsugane S; Yamamoto H; Miyajima N; Toyoshima K; Yamamoto T; Yokota J; +
CS Pathology Division, National Cancer Center Research Institute, Tokyo, Japan.
SO CANCER RESEARCH, **(1989 Jun 1)** 49 (11) 3104-8.
Journal code: 2984705R. ISSN: 0008-5472.
CY United States
DT Journal; Article; (JOURNAL ARTICLE)
LA English
FS Priority Journals
EM 198906
ED Entered STN: 19900306
Last Updated on STN: 20000303
Entered Medline: 19890623
AB The incidence and association with 10-year survival of **amplification** in five protooncogenes or transforming genes were retrospectively examined using DNAs extracted from formalin-fixed, paraffin-embedded blocks of tissues obtained from 176 consecutive patients surgically treated for primary **breast carcinoma**. The incidences of greater than threefold **amplification** of hst-1, int-2, c-erbB-2, ear-1 (one of c-erbA), and c-myc were 12, 13, 16, 10, and 4.0%, respectively. hst-1 and int-2 were almost always coamplified (21/22), while c-erbB-2 and ear-1 were frequently coamplified (18/28) with almost the same copy number. The hst-1 and int-2 pair and the c-erbB-2 and ear-1 pair, localized on chromosomes 11q13 and **17q21**-22, respectively, in normal cells, were inferred to be constituents of different **amplification** units. **Amplification** of hst-1 and/or int-2 was detected preferentially in the younger age group, and was correlated with poorer prognosis in cases carrying four or more copies of the genes. **Amplification** of c-erbB-2 and/or ear-1 was strongly correlated with poor prognosis in all 176 patients, especially those with lymph node metastasis. **Amplification** of c-myc was also correlated with poor prognosis. Cox's life-table regression analysis showed that **amplification** of c-erbB-2 had a prognostic value, which was independent of other known prognostic factors such as lymph node status and tumor size.

L5 ANSWER 4 OF 5 MEDLINE DUPLICATE 4
AN 90028019 MEDLINE
DN 90028019 PubMed ID: 2572268
TI c-erbB-2/c-erbA co-**amplification** indicative of lymph node metastasis, and c-myc **amplification** of high **tumour** grade, in human **breast carcinoma**.
AU Tavassoli M; Quirke P; Farzaneh F; Lock N J; Mayne L V; Kirkham N